*THE THERMOSTATIC CONTROL OF HUMAN METABOLIC HEAT PRODUCTION**

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The fact of human thermal homeostasis in warm or cold environments, at rest or under maximal activity and heat production, has stimulated numerous attempts to clarify its various aspects. Although such efforts have been made since 1885 or earlier, no integrated set of experimental facts has been forthcoming to explain it in terms of measurable effects and causes. Effects—responses by augmented metabolic heat production, sweating, or vasodilation—have not been demonstrated until recently[1] to be quantitatively and inseparably related to causes: stimuli of temperature at specific terminal receptor sites of our nervous system. Until such a causal analysis is accomplished, the homeostatic system cannot be considered clarified.

Lately, the introduction of gradient calorimetry[2, 3] (for measurements of the physiological responses) and cranial thermometry[4] (for measurements of the stimulus at the thermostatically controlled interior of the body) have sufficiently raised the power of resolution, which the instruments must have to solve their task. Moreover, simple ways have been devised to separate cutaneous from central temperatures. Thereby, true correlations between a response and either one of the two stimulant receptor temperatures under consideration are readily distinguished from false, coincidental correlations. (False correlations between a response and one of the two stimuli obtain as long as cutaneous and internal temperatures are themselves interrelated under physiological conditions without experimental interference.)

With these precautions against fallacious reasoning and with the refinements of methods as outlined above, an experimental resolution of "physical" heat regulation in man was first obtained and presented in these Proceedings[1] and elsewhere.[4, 5] (After these results were published, observations were extended to environments of 50° and 55°C and again no driving influences of skin thermoreception could be found. By cooling the skin below the threshold of cold receptor action, to 32°C (and less), however, it was possible to reduce the rate of sweating. It is as yet unknown whether this influence upon sweat glands is direct, or a consequence of vasoconstriction, or perhaps induced by cold reception.) Subsequently, the resolution of the combined "physical" and "chemical" mechanisms was carried out as follows.

Results.—In Figure 1, as a result of three months of experimentation with one healthy young man, rates of metabolic heat production in response to cold, and rates of sweating heat loss in response to heat, are plotted against cranial internal temperatures, measured at the tympanic membrane of the ear. Cold-stimulation was obtained in water baths; warm-stimulation, in the gradient calorimeter at high environmental temperatures. Coincidental correlations were deliberately disrupted through measures driving skin and central temperatures apart. Nevertheless, the deviations hardly exceeded the experimental errors and the plot is mean-

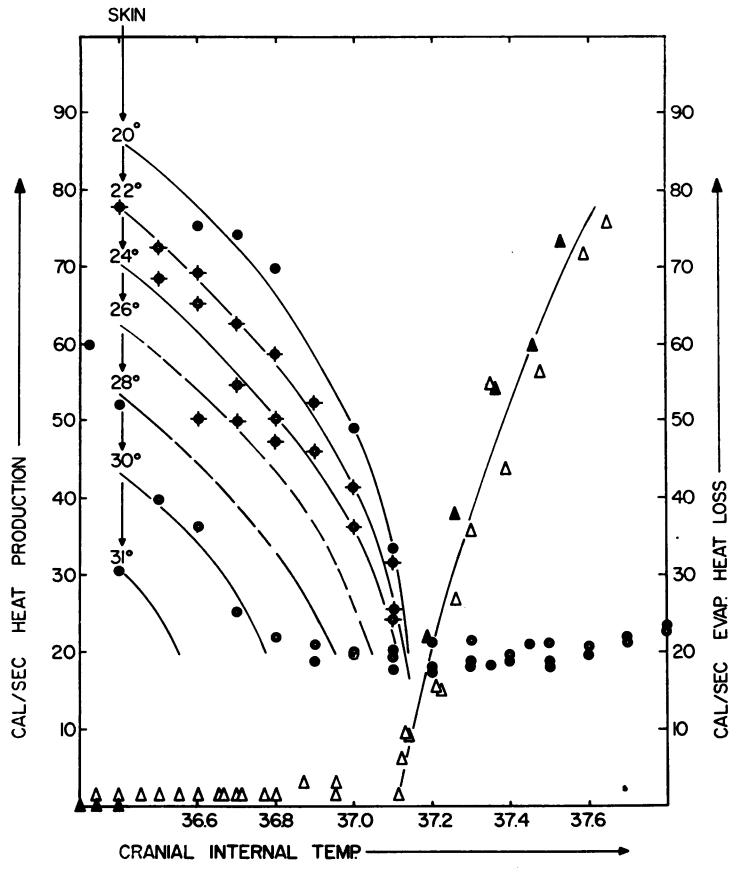

FIG. 1.—Experimental Resolution of "Chemical" and "Physical" Temperature Regulation. Thermoregulatory heat production (abscissae, circles, cal/sec) at low and constant cranial internal temperature (ordinates) is determined by steady cold-stimulation of skin (temperatures 31° to 20°C). Central warm-stimulation (ordinates) leads to depressing counteraction, which becomes complete at individual "setpoint of thermostat," 37.1°C. Thermoregulatory sweating (triangles, abscissae) is uniquely determined by internal sensory warm-reception. It begins at setpoint, rises to comparable evaporative loss (cal/sec). Result is tenacious maintenance of setpoint (homeostasis) over fourfold range of production or losses. *Note:* For narrow temperature range on ordinate, resolving power of classical methods was insufficient.

ingful on either side of a sharply determined "setpoint of the human thermostat" (at 37.11°C internal cranial temperature for this subject). The tenacious maintenance of a "setpoint temperature"—that is, the fact of human thermal homeostasis—is quantitatively explained as follows.

In response to minute changes with increasing *central* temperature, evaporative heat loss by sweating rises to extraordinary magnitude on the right side of the setpoint. Here the strict relation with cranial internal temperature is unaffected by the variations of *skin* temperature between 32° and 37°C that had been artificially introduced. On the left side, metabolic heat production rises to comparable values as internal temperature is reduced below the setpoint in minute changes on

this narrow scale. The magnitude of that particular response, however, is clearly dependent on whatever skin temperature prevailed, as indicated by the lines connecting measurements taken at any individual one of the various skin temperatures between 31° and 20°C.

It followed that "chemical" heat regulation by increased metabolic heat production was determined by both peripheral (skin) as well as central temperature, or thermoreception. It followed also that in "physical" temperature regulation by sweat-gland activity, *central* thermoreception was the origin of the driving warm-impulses.

In other words, in chemical heat regulation (controlling production), the central component acted as a synaptic station relaying and modifying afferent sensory impulses from the skin. By contrast, in physical heat regulation (controlling loss), the central component acted like a terminal receptor organ with first neurons, by showing the unique capability of translating minute changes of temperature, as stimuli, into nervous activity mediating powerful responses.

This puzzling contradiction will be explained later in our discussion with the existence, proven by classical authors, of two "centers" with fundamentally different characteristics. Additional experimental evidence shall first be introduced concerning the nature of the neural cold-messages from the skin, which Figure 1 so clearly demonstrates. By rapid and continuous recording of human oxygen consumption with new methods[6, 7] we have been able to show in various independent ways that the sensory messages driving thermoregulatory heat production in response to cold are a result of nerve impulses of increasing frequency from thermoreceptive nerve endings at the skin as investigated by Hensel and Zottermann.[8]

1. By transient application of the stimulus of cold to the skin in a water bath, in the absence of measurable changes of internal cranial temperature, the metabolic rate of man may be deliberately raised to, and lowered from, a rate of 3 to 4 times basal oxygen consumption.

2. To sudden lowering of total skin temperature, the rate of oxygen consumption responds with a transitory, overshooting "spike" and settles at a new and constant higher rate, commensurate with the new and lower temperature level. The frequency of electrical discharges from isolated thermoreceptive fibers of the cat in experiments of Hensel and Zottermann[8] responded in the same manner.

3. To sudden elevation of skin temperature, the metabolic rate of man responds with an overshooting and transitory depression of thermoregulatory heat production. The frequency of electrical discharges from isolated thermoreceptive fibers of the cat responded likewise.[8]

4. When the effects of changes in skin temperature are excluded, and only such measurements of metabolic rate and steady skin temperature are considered at which the cranial internal temperature was the same, for example 36.6°C, as in Figure 2*a*, a reproducible, peculiar relationship appears: metabolic rate first rises and then falls when skin temperature is brought below a critical level of maximal thermoreceptive response. The frequency of electrical discharges from isolated thermoreceptive fibers in ref. 8 followed the same pattern (Fig. 2*b*).

The extended series of experiments, from which only those performed at one certain internal cranial temperature, 36.6°C, had been selected for Figure 2*a*, is fully shown in Figure 2*c*. In this representation, the effect of skin temperature

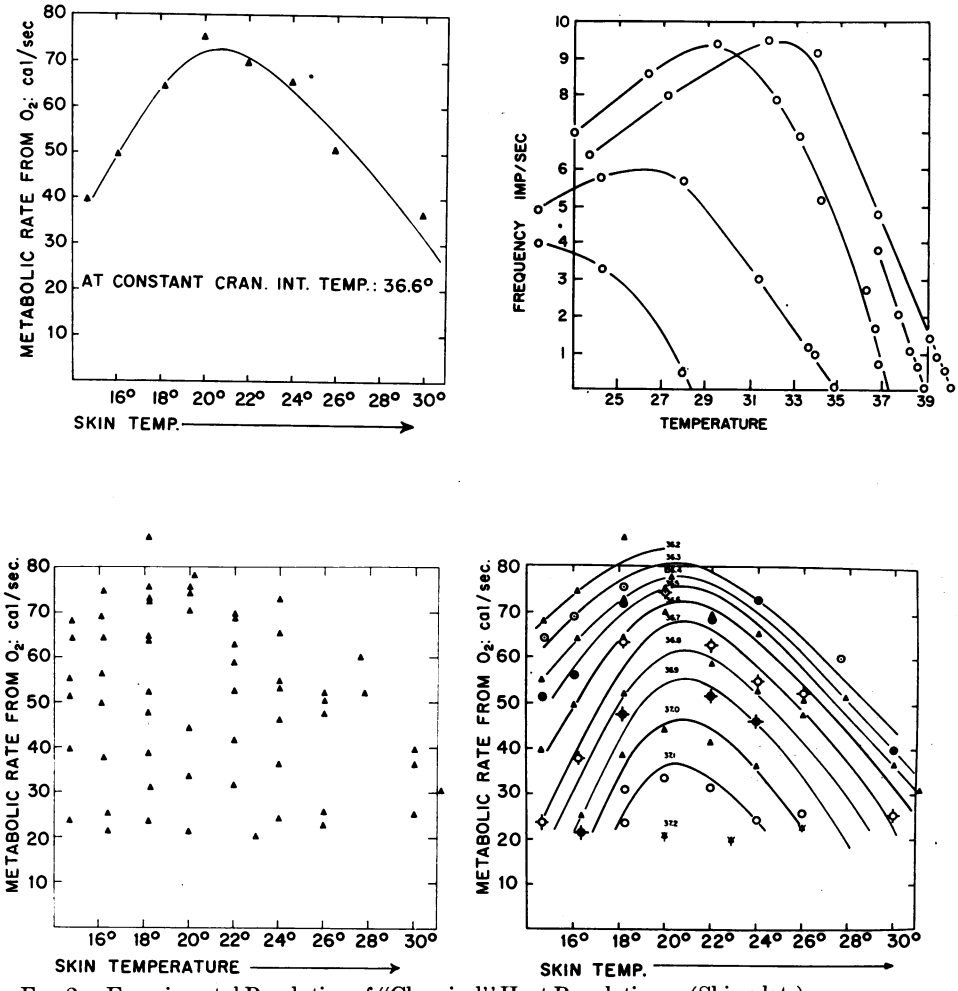

FIG. 2.—Experimental Resolution of "Chemical" Heat Regulation. (Skin-plot.)
(*a*) Metabolic response to steady cold-stimulation of skin (observed at *constant* central temperature, 36.6°C) attains maximum and then declines.
(*b*) Electrical discharge frequency from isolated thermoreceptive fibers in cat's tongue (after Hensel and Zottermann[8]).
(*c*) Metabolic rates plotted against skin-temperatures at *varied* cranial internal temperature yield seemingly senseless plot.
(*d*) Interconnection of measurements obtained at any one of many cranial internal temperatures in Figure 2*c* with "best-lines" yields Figure 2*d*, a family of curves, the resolution of "chemical" heat regulation. Parameters discussed in text.

upon the rate of oxygen consumption appears to be wholly obscured by another factor so that a senseless plot results.

This factor is the influence of cranial internal temperature, which had been deliberately and arbitrarily varied in the series of measurements. For when, as in Figure 2*a*, all measurements obtained at one and the same internal cranial temperature were interconnected with a common "best line," the senseless field of scattered points was converted into a family of curves and a complete and quantitative resolution of "chemical" heat regulation was obtained for this individual (Fig. 2*d*). This representation is different from, though equivalent to, the one in Figure 1,

which is based on the same observations. Either one of the two plots permits us to predict what the rate of thermoregulatory heat production would be in this individual for any combination of internal cranial and cutaneous temperatures. From Figure 2*d* some important parameters may be read without further explanation.

The threshold of skin thermoreceptor excitation was near 33°C (extrapolated to the right). Maximum excitation took place at 20–21°C skin temperature. The largest possible response was approximately 350 per cent of resting metabolic rate. Response-sensitivity between the threshold and the maximum was roughly 1 cal/sec heat production to each 0.2°C of skin temperature reduction. The depressing action of rising cranial internal temperature was of the order 1 cal/sec for each 0.01°C. Depression became complete between 37.2° and 37.0°C of central temperature, i.e. at the setpoint of this individual for the onset of sweat-gland action.

Interpretation.—A thermostatic neural mechanism, as described by Figure 1 in quantitative terms of stimuli and responses, cannot be anatomically organized in arbitrary ways. It must be based upon a certain minimum number of effector, central, and receptor components, interconnected as shown in Figure 3. (For the present purpose of interpretation, the background drawing of the brain in Figure 3 should be disregarded. While the calorimetric analysis demonstrates the existence of the components drawn with bold lines, the actual location of corresponding centers and pathways will be discussed only later with reference to classical discoveries of experimental surgery.)

a. A "heat *maintenance* center," *P*, receives from cold-receptive nerve endings of the skin through an afferent pathway, *sk*, afferent impulses for thermoregulatory heat production, which is initiated and maintained through an efferent pathway, *m*, in the metabolizing tissues of the effector organs.

b. A "heat *loss* center," *A*, without receiving warm-impulses from the skin, must translate the stimulus of temperature in its own cells into nervous impulses which initiate and maintain the responses of sweating and peripheral vasodilation through the efferent pathways *sw* and *v*.

c. Through a third efferent pathway, *d*, the depression of thermoregulatory heat production must be effected by sensory impulses from an internal receptor site, such as *A*, through connections with effector neurons of the "heat maintenance center," *P*.

In summary, center *P* functions simply as a synaptic relay station with one efferent and two afferent pathways. Center *A* functions for temperature like any other terminal receptor organ with first neurons for whatever quantity it "senses." Center *A* is active by way of three efferent pathways upon the three unconscious thermoregulatory functions.

This type of system satisfies the experimental facts thus far revealed by calorimetry and experimental surgery, while it does not invoke any speculative assumptions. It cannot, of course, rule out conclusively such speculative assumptions as have been occasionally entertained; for example, the existence of additional central thermoreceptive organs having efferent pathways, afferent to the effector neurons for sweating and vasodilation. Such organs have been assumed by some to exist elsewhere in the brain or in the large central vessels or even in the cranial mucous membranes where our measurements were carried out. There is, however, one hypothesis based on serious experimental observations: the possible existence of an

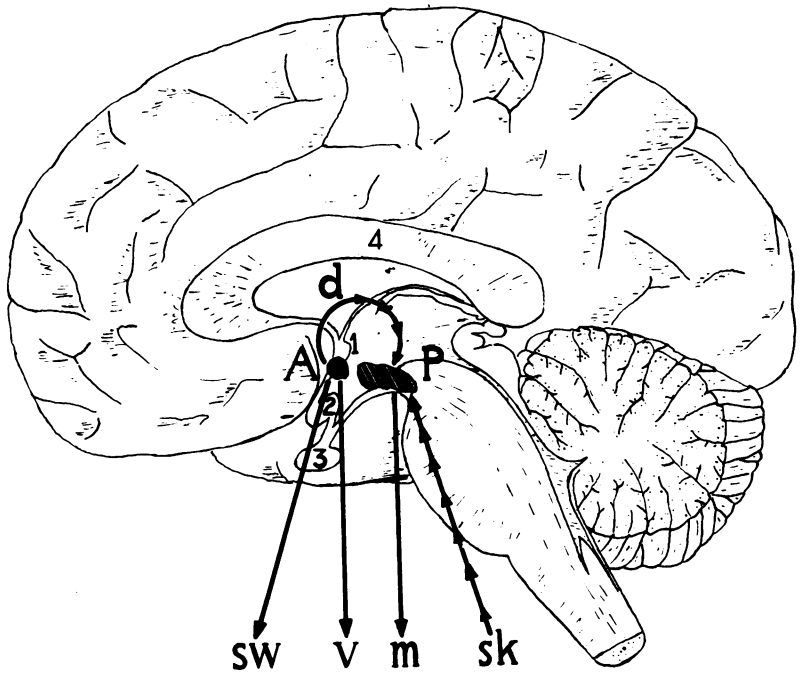

FIG. 3.—Minimum number, interconnections, and characteristics of thermoregulatory centers to be postulated from calorimetric evidence on man are shown with bold lines. Anatomical location of centers established by classical experimental neurosurgery on animals is shown by superimposition upon independent background drawing. Centers as shown have the characteristics required for the following functions: In the first place, sweat glands and cutaneous vessels (through efferent pathways *sw* and *v*) are operated from an *internal*-sensory receptor site, *D*. Secondly, metabolizing tissues (through pathway *m*) are operated for chemical heat regulation from *skin* as terminal receptor site (with afferent pathway, *sk*). Thirdly, metabolic response to increased frequency of cold-impulses in afferent pathway, *sk*, is counteracted and depressed by warm-impulses through another afferent pathway, *d*, originating from an *internal* thermoreceptive site such as *D*. Identity of one center, *D*, as the origin of both (depression of metabolic responses and operation of *sw* and *v*) is documented by references 17 and 18. So is *thermal indifference of center P*. Correct localization on background drawing, of centers *D* and *P* in anterior and posterior hypothalamus is documented by reference 9–17, 21–27, 29–35, and 38. Pathway *d* is anatomically identified by Hemingway,[33] upper part of pathway *sk*, by Anderson and Berry,[3] and *m* by Hemingway,[13–11] and Keller,[11] Anatomical "landmarks": (1) Commissura anterior, (2) Chiasma opticum, (3) Hypophysis, (4) Corpus callosum.

additional mechanism based on cold-receptor cells in the septum pellucidum.[38, 39] This mechanism might perhaps be active in deep hypothermia and support to a certain degree the major mechanisms described above.

Discussion.—Whereas the mere existence of a minimum of components, as shown in Figure 3, appears to be established with the calorimetric evidence of Figure 1, only experimental neuroanatomy or neurosurgery can ultimately localize these components within our central and peripheral nervous system. Such evidence in fact exists already and shall be discussed as follows:

1. The afferent pathway *sk* from the skin has been demonstrated by Anderson and Berry[9] who found in cats, after transection of the thoracic spinal cord, degenerating fibers from the lateral spinothalamic (Gowers') tract ascending into

and terminating within the subthalamus and posterolateral hypothalamic regions.

2. A "heat maintenance center," P, was delineated in the *posterior* hypothalamic gray as early as 1912 by Isenschmidt and Krehl[10] in experiments with ablation of dispensable tissue, and in 1914 by Isenschmidt and Schnitzler[11] by determination of the minimum lesion required to abolish shivering. Keller and Hare[12] have shown that after destruction of center P the heat loss functions (of center A) remain intact. Electrophysiologically, the function of center P has been established with electrical stimulation by Birzis and Hemingway[13] and the efferent pathway, m, has been determined by the same author with lesions,[14] electrical stimulation,[13] and with action-current recordings on shivering dogs.[15] Keller[16] has shown the continuation of pathway m in the pyramidal bundles.

3. Most important, the *indifference of center P to thermal stimulation* has been demonstrated independently by Hemingway *et al.*[17] with a diathermy electrode at the basal surface of the brain *near* the center, and by Freeman and Davis[18] using a diathermy electrode *within* the center. With the combined evidence of calorimetry and neurosurgery, center P in the posterior hypothalamus is therefore identified as a *synaptic* center relaying peripheral afferent impulses and incapable of responding itself to the stimulus of temperature. These characteristics differ fundamentally from those of center A.

4. The existence of a "heat loss center," A, in the *anterior* hypothalamus, was discovered by Aronsohn and Sachs in 1885.[19] It was delineated with improving precision by Barbour,[20] Ranson and Magoun,[21] Ranson,[22] Magoun *et al.*,[23] Hess and Stoll,[24] Folkow *et al.*,[25] and Anderson *et al.*[26] Ranson and Teague[27] have shown that its destruction leaves the "heat maintenance" functions intact, or even excessively responding (Pinkston, Bard, and Rioch[28]). Destruction of center A *eliminates* heat loss responses (Clark, Magoun, and Ranson[29]). Electrical stimulation of A *elicits* heat loss responses, causes the cessation of shivering (Hemingway *et al.*[30]), and thereby produces deep hypothermia (Andersson and Persson[31]). On patients, hyperthermia has been observed by Gagel[32] when center A was destroyed by neoplasm. While details in identification of pathways *sw* and *v* seem to require more extensive study, their existence is generally accepted.

5. Efferent pathway d, responsible for the central depression and thereby the control of shivering when temperature at center A increases, has been directly established by Hemingway[33] with the recording of action currents during electrical stimulation of the "heat loss center," A.

6. Most important, *thermal* (not only electrical) stimulation of center A has been shown by Magoun *et al.*[23] and by Stroem[34] to produce the heat loss responses. This phenomenon and, in addition, the cessation of shivering upon warm-stimulation of center A was shown by Hemingway[17] and by Freeman and Davis[18] independently. These authors applied the stimulus of heat selectively to center A while minimizing its effect upon center P, to find the origin of shivering depression.

7. Ultimately, Curt von Euler[35] has observed slow action potentials, commensurate with the artificially applied stimulus of temperature at center A, indicative, as he observes, of thermoreceptor cells. These findings are comparable to those of Svaetichin and MacNichol[36] who recorded slow potentials commensurate with the stimulus of light, near the optical receptor structures, first neurons of the retina. Another pertinent example are slow potentials commensurate with CO_2 tension and

respiratory volume in the respiratory centers of the medulla recorded by von Euler and Soederberg.[37]

In summary the neurosurgical evidence as quoted and the calorimetric evidence reported here are mutually consistent and confirmatory. They tend to establish the characteristics of a terminal sensory organ (concerned with warm-reception) for the anterior hypothalamic "heat loss center" designated "A" in Figure 3.

To understand the fact of human thermal homeostasis on the basis of the findings (Fig. 1) it remains to discuss briefly why conscious sensations and willful thermoregulatory actions of man must first provide the proper preconditioning. Too often the external conditions or workloads would demand a rate of heat loss so excessive that even the maxima of sweating and peripheral blood flow could not meet it. Also the air may be too humid to permit complete evaporation of the sweat-gland product. A cool environment may force internal temperature below the setpoint. These extremes, under which the system described in Figure 1 could not possibly function, are avoided by "Pavlovian" action. For one example at skin temperatures of 31°C the gap between the desired setpoint, 37.1°C (where oxygen consumption *should* begin to rise) and the intersect of the 31°C line at which it actually would (36.6°C), is bridged by the Pavlovian measure of voluntary muscular exertion. Iberall[40] has found that people warm themselves by deliberate movements whenever skin temperature drops below 34–33°C. Similarly, in other environments, conscious sensations of temperature will lead to voluntary locomotion into a more comfortable environment, to application or removal of clothing, to changes of bodily posture and radiating surface, to immersion in warm or cool water, to voluntary exercise, to building a shelter against cold or for shade, and ultimately to artificial heating or cooling with external sources of energy. Thus, autonomic and Pavlovian measures are equally important in their own ways; the first in providing for the range of human temperature control, the latter for its almost unbelievable precision.

In view of constructive comments on conclusions drawn in references 1, 4, 5, and here, the authors wish to state that they do not disregard the existence of additional thermoregulatory activity at segmental levels nor certain direct influences of skin temperature upon sweat glands or vessels. In the vascular system those additional factors were observed with our methods, and discussed in references 1 and 4. In the sudomotor system and in shivering they are perhaps too small to be detected as long as the powerful hypothalamic system is in operation.

Summary.—By cranial thermometry combined with indirect and direct calorimetry, the two mechanisms of human "chemical" and "physical" temperature regulation have been experimentally resolved in terms of metabolic, sudomotor, and vasomotor responses of reproducible magnitude to the degree of cold-stimulation at thermoreceptive nerve endings of the skin and warm-stimulation of an extremely sensitive, centrally located thermoreceptive organ. The simplest possible neuroanatomical basis for explanation of the facts observed has been discussed with reference to the findings of classical experimental neurosurgery. It is concluded that the function of the "heat maintenance center" in the posterior hypothalamus is synaptic, relaying activity of increasing frequency from thermoreceptive endings in the skin for increasing response by metabolic heat production. This center is indifferent to the stimulus of temperature. The "heat loss center" in the anterior

hypothalamus, however, acts as a terminal receptor organ for temperature, comparable to the retina, the anatomically related receptor organ for light. This organ controls not only the direct responses to heat by vasodilatation and sweating but also through a third efferent pathway, afferent to the "heat maintenance center," it indirectly controls, with counteracting impulses, the response by metabolic heat production to cold-stimulation of the skin. The result of this threefold function is a thermostatic performance of astonishing power and precision.

Navy Hospital Corpsmen D. L. Drake and F. D. Crabill and, on several occasions, Ensign C. Chestnut or one of the authors volunteered for the extended experiments with exposure or immersion. Corpsman R. D. Arrieta underwent one exceptionally rigorous test. The expert technical assistance of Mr. G. W. Newlon and Mr. L. R. Younkins is gratefully acknowledged.

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*COCHLEAR POTENTIALS IN THE MARMOSET**

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From a comparative standpoint the primate ear claims a special interest, for this order is the one to which man belongs. It is more than a little surprising, therefore, that our knowledge about the hearing of members of this group is severely limited. Apart from man himself, only the chimpanzee and Old World monkeys (the rhesus monkey and its close relatives) have been tested for auditory acuity.

These considerations led Seiden,[1] working in our laboratory, to carry out auditory measurements in the marmoset, one of the New World primates that is considered more primitive than the species mentioned above. He used five animals of the species *Hapale jacchus* and trained them to respond to sounds by a shock avoidance method. The animal was placed in a tipping cage and was required to cross over its center line when a tone was sounded, being given a mild electric shock if it failed to do so within 0.5 to 3 seconds after the tone began. Observations of threshold sensitivity were attempted over a frequency range from 100–80,000 cycles, but all the animals reached their limit between 25,000 and 37,000 cycles.

After these measurements had been completed, the same animals were used for cochlear potential studies, and the results of these studies are the subject of the present report.

The animals were anesthetized with diallylbarbituric acid and ethyl carbamate (Dial), in a dosage of 1 cc/kg of body weight. The recording electrode was a platinum foil on the round window membrane, with an indifferent electrode in inactive tissue nearby. In all the animals the observations were made on the right ear. The stimulating and recording equipment described earlier in our experiments on the cat[2] was used to obtain measurements over the range of 100–100,000 cycles.

Some of the results are given by the dashed-line curves of Figures 1 and 2, together with Seiden's curves of threshold acuity shown by solid lines. Figure 1 shows a representative ear, and the data for three other animals were closely similar to these. As will be seen, the potentials were obtained over the full range of 100–100,000 cycles. As the frequency was raised above 100 cycles, the sensitivity in terms of these potentials improved slowly, except for an irregularity around 700 cycles, until 1,500 cycles was reached, after which the level remained fairly uniform up to 20,000 cycles. Beyond this point the sensitivity was poorer, and the course of the curve was somewhat more irregular than elsewhere.

A comparison with the behavioral threshold curve shows a number of differences. The gain in sensitivity as the frequency is raised above 100 cycles is noticeably